PHSB STUDIES

A Special Report Series by the N.C. Department of Human Resources, Division of Health Services, Public Health Statistics Branch, P.O. Box 2091, Raleigh, N.C.

No. 14 March 1979

ASSOCIATION OF INFLUENZA WITH CONGENITAL TRACHEOESOPHAGEAL FISTULA AND OESOPHAGEAL ATRESIA: AN ANALYSIS OF CLUSTERS

Epidemiologic Patterns of the Malformation

Research and management programs have had notable successes in solving many problems associated with birth defects; however, the origin or causes of several defects remain a mystery. Congenital tracheoesophageal fistula (TEF) and oesophageal atresia (OA) are two such conditions whose etiology is not understood.

TEF is an abnormal passage between the trachea and the esophagus. It assumes any of a large number of possible forms and is often characterized by a spectrum of symptoms including choking, problems with swallowing and even death in a large percentage of cases. OA is a congenital lack of continuity of the esophagus and is characterized by excessive salivation, gagging, vomiting, cyanosis and dyspnea. TEF and OA often appear together and are often studied together as though they were a single entity. We shall follow this precedent if for no other reason than to amass enough cases.

Knox (1,2) presented some data that suggest that cases of TEF and OA occur in clusters over time. By considering the monthly and annual number of cases admitted to hospitals in Birmingham, England from 1950 to 1955 and in the Newcastle region from 1950 to 1958, one finds years in which the incidence of reported cases was far in excess of that of the surrounding years. However, it is less clear that a particular month repeatedly was associated with a high reported rate. Babbott and Ingalls (3) found a similar pattern in Pennsylvania County for the period 1951 through 1958, despite relatively uniform birth rates. Koop (4), in his experience with over 300 infants treated for OA in a 15-year period in Philadelphia, found that cases of OA were admitted fairly consistently over the years in bunches in April; this suggests a seasonal effect. He reported that on one occasion in a period of 21 days, 17 infants with OA were admitted to the Neonatal Unit and that over half of these came from the same area of Pennsylvania countryside. Other physicians have reported to us that the incidence of OA and TEF seems to be greater in April or May. Also, see the paper by Slater et al (5).

Contrary to this, there have been reports of the absence of clustering in time or space of OA. There are indeed times and places where clustering is absent but this could simply reflect that a sporadically-behaved cause is absent as is true of infectious diseases or it could imply an insensitive data-collecting mechanism. For example, we have found that data from death certificates regarding certain malformations suggest disparate conclusions about patterns of the disease depending on whether the condition is listed as the underlying cause of death or simply mentioned at all.

Also, it is possible that a direct link may be found between a specific malformation as reflected in data comprised of underlying causes of death (with no or few other conditions mentioned) and a specific environmental factor (e.g., influenza) and, further, that this link may be obscured by including in the data deaths for which the malformation is simply mentioned. Conceivably, such a mention may be only a small part of a constellation of several anomalies associated with the infant, a constellation caused by different factors (e.g., thalidomide, rubella).